Telephone: MAYFAIR 0282.
Telegraphic Address: LITHOTRITE [illegible]

JOHN W[illegible]
267, Oxford Street,

Instrument Makers to the Royal [illegible]
and to the principal Hospitals [illegible]

CONTRACTORS TO H.M. GOVERNMENT
Special Orders and Repairs executed with promptitude

OPERATION & PROJECTION LAMP

(Prov. Patent No. 26705)

As an operation lamp the above is particularly suitable for ophthalmic work, as an almost red-free light of 40 c.p., without shadows, can be obtained.

For examination of the fundus a filter can be supplied which cuts out all red rays.

The tube is provided with a slotted opening to accept 1" microscopic slides for projection on to a screen. If wider (2") slides are to be used a special adapter with a spring gate is supplied in which the slide can be held or moved. This, with a slightly different optical system and an iris diaphragm (which can be operated), gives a magnification of 50 diameters at 10 feet.

A transilluminating cone and slides for testing for colour blindness can also be supplied.

Prices from £7 10 0. Full particulars on application.

While going to press, we learn with profound sorrow of the death, after his long illness, of SYDNEY HARRY STEPHENSON, M.B., F.R.C.S.(Edin.), D.O.(Oxon), Editor of *The British Journal of Ophthalmology*. We shall publish an account of his life and work in our next number.

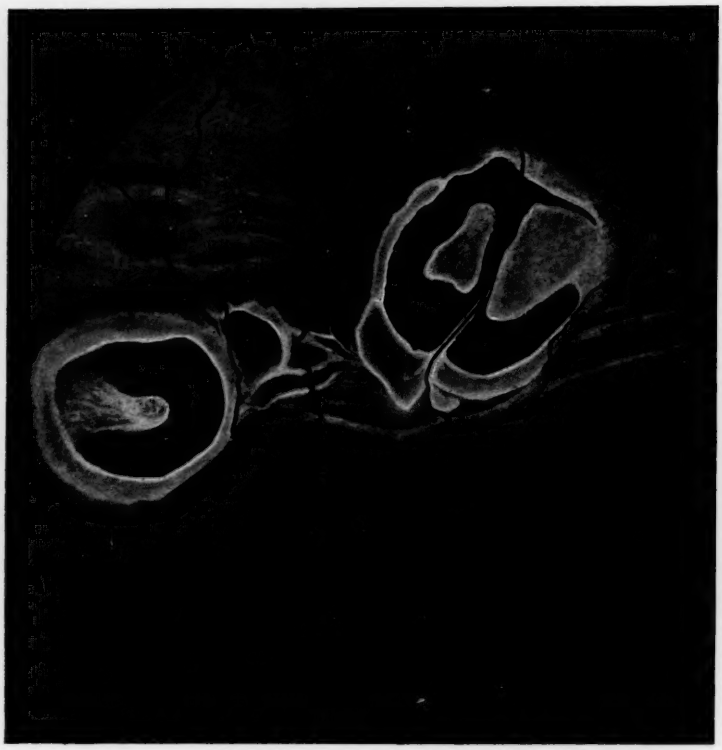


PLATE I.

Holes in a detached retina.

To illustrate Sir William Lister's paper on "Holes in the Retina and their Clinical Significance."

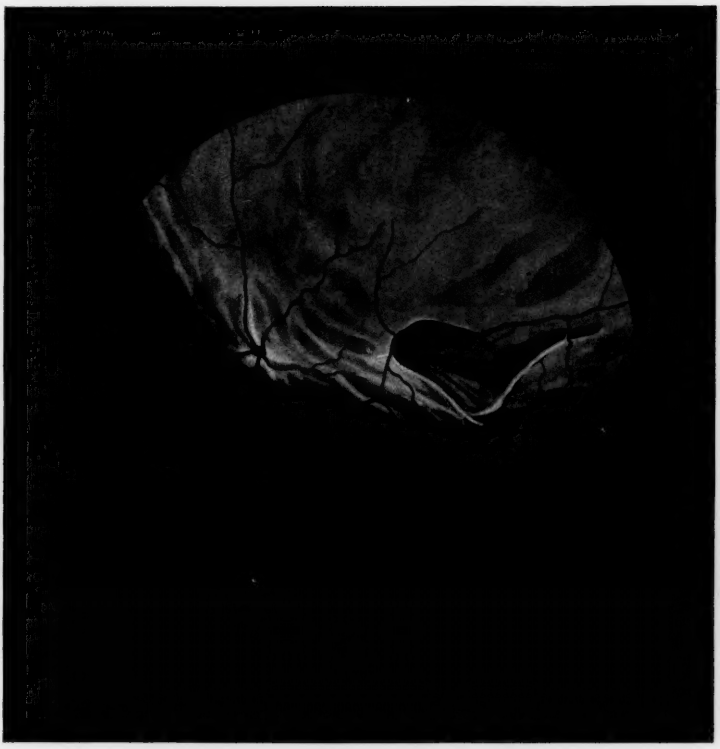


PLATE II.

Globular detachment above in which there is a large retinal rent, through which not only the choroid but also outer surface of retina is visible.

To illustrate Sir William Lister's paper on "Holes in the Retina and their Clinical Significance."

THE BRITISH JOURNAL

OF

OPHTHALMOLOGY

JANUARY, 1924

COMMUNICATIONS

HOLES IN THE RETINA AND THEIR CLINICAL SIGNIFICANCE*

BY

SIR WILLIAM LISTER

LONDON

I HAVE chosen as the subject of my paper, one on which I fear I cannot throw much new light, but it is one to which, in my opinion, too little attention is paid clinically. My subject is "Holes in the Retina and their Clinical Significance," and my remarks will be divided under three headings:—

I. The various clinical varieties of holes in the retina.

II. Their aetiology.

III. Their clinical significance.

I. Clinical varieties

For convenience we may divide holes in the retina into two main groups—macular holes and peripheral holes, and subdivide each of these under the two headings of traumatic and non-traumatic.

(1) ***Macular holes.*** We are all familiar with the so-called "macular holes"; they occur both after trauma and without trauma.

(a) ***Traumatic.*** The greater number of macular holes follow a blow on the anterior part of the eye from non-perforating missiles,

*Delivered before the Ophthalmic Section of the Academy of Medicine, Toronto, October, 1923.

6-17-25
amw

such as stones, fists, etc. These cases have been well described and illustrated by various observers.

As seen with the ophthalmoscope, the hole may be single, round or oval, or there may be two or more holes. The hole has a sharply punched out appearance, its margin being whiter than the surrounding retina and occasionally decorated with small rounded masses. The edge is sometimes raised, indicating a slight detachment, but any great degree of separation is very rare. The centre of the hole is bright red and frequently exhibits some pigmentary disturbance or glistening dots.

(b) *Non-traumatic.* The appearance of a hole at the macula is also found in cases where no history of a blow is obtainable, and in which retino-choroidal degeneration is the most probable cause. Whether these are true holes or whether the appearance of a hole is due to cystic degeneration of the retina will be discussed later.

(2) *Peripheral holes.* (a) *Traumatic.* We have all seen cases of holes situated in the periphery of a detached retina, following a blow. These are large oval or circular rents or they may be slit-like or shaped like a horse-shoe and in many of the latter the central portion is bent inwards. Occasionally large gaps in the retina are found situated so far forward that only the posterior margin of the rent is visible. These are probably due to tearing of the retina from the ora serrata. Traumatic peripheral holes are usually associated with considerable detachment.

In my own experience peripheral holes due to trauma without detachment are very uncommon. I saw one case some years ago, that of a boy who was struck by a piece of paving material on the outer side of the sclerotic, sufficiently hard to make the eye bleed. His sight was impaired for a few days but later full acuity was restored. It was only after dilating the pupil and examining the extreme periphery that a hole in the retina was discovered on the outer side. It was so far forward that only the posterior margin of the rent was visible, the anterior margin being shut off from observation by the edge of the pupil and thus very possibly the tear in this case was at the ora serrata.

The portion of the edge of the rent which could be seen was white and slightly raised and through the gap the choroid was seen a bright red; there was no visible detachment of the retina and the field was full. This case had a special interest from the medico-legal aspect, for the boy was on duty when he was struck and was therefore entitled to compensation for injury. His sight, however, was perfect and therefore at the time compensation only for loss of work could be claimed. On the other hand, with this hole, detachment might occur at any moment which would have caused

complete loss of sight in this eye, when he would have been entitled to a heavy compensation. I have lately tried to trace this boy to learn the present condition, but failed as he had gone to New Zealand.

(b) *Non-traumatic.* Holes associated with choroiditis or contraction of the vitreous are according to my experience more frequently discovered during pathological investigation than with the ophthalmoscope.

II. Aetiology of Holes

There are some who have suggested that in every detachment there is a hole in the retina. Such a view can hardly be accepted when the different consistency of the fluid in the inter-retinal space is noted in different cases.

Though in some cases of detachment, where no hole is discoverable, the consistence of the inter-retinal fluid is clear and thin after fixation and similar in appearance to that of the degenerated vitreous, yet I think we shall all agree that in the majority of cases of detachment without a hole, the inter-retinal fluid after fixation sets into a firm, jelly-like mass. On the other hand, in *every* case in which a hole has been found, the consistency of the inter-retinal fluid after fixation is exactly similar to that of the fluid vitreous. If the fluid in the inter-retinal space has free communication with the vitreous by means of a hole it would seem incredible that the two should not mix. One is, therefore, forced to hold the view that when, after fixation, a solid coagulum is found in the inter-retinal space, there cannot be a hole.

The cases where the inter-retinal fluid is of similar consistency to the vitreous and no hole is found, are probably either (1) those in which the observation is incomplete, the hole being obscured by a fold in the retina or so small that it is undetected, or (2) those in which a change has taken place in the proteid molecules of the inter-retinal fluid, breaking this up into more readily diffusible substances.

From the variety of the position of holes and of their associated conditions, it would be inferred that there must be several different causes which bring them about.

Traumatic Macular Holes. A considerable amount of literature has appeared on the cases of so-called macular holes following trauma giving reports of the pathological investigations. That actual holes occur at the macula is proved by their having been found microscopically. Whether these holes were caused directly by the blow or whether the retina had first undergone a cystic degeneration as the result of a blow and the hole was caused subsequently by the rupture of the cyst walls is as yet uncertain.

Cystic degeneration is frequently seen in uninjured eyes in the forward portion of the retina near the ora serrata, and, though less common in the central region, large cysts at the macula with very thin walls are occasionally met with. It has yet to be determined whether all cases in which the ophthalmoscope reveals the appearance of a hole have definite gaps in the retina, or whether in some the ophthalmoscopic appearance of a hole may be brought about by a cyst of the retina alone without rupture.

Unfortunately the justification for removing eyes with the ophthalmoscopic appearance of a macular hole is so rare that little opportunity is offered for enlarging our knowledge.

A reasonable explanation of this macular condition would seem to be, that since most macular holes are due to a blow on the anterior part of the eye, the macula will lie at the point of contrecoup. Not only is the macula the most delicate part of the retina but it is at this spot that the rending force of the pressure waves starting from the site of impact will be most felt, as I have described in a paper on ruptures of the sclerotic at the point of contrecoup. This rending force in some eyes may cause an actual rupture of the retina, in others it may be followed by cystic degeneration, and, in a proportion of these cases rupture of the cyst walls takes place.

Macular holes are, however, also seen as a result of lateral blows on the eye. These were seen during the war in a certain number of cases, where missiles passed through the side of the orbit without actually striking the eye. With these injuries it was the rule to find the concussion changes situated adjacent to the site of impact, but in some cases changes were also found at the macula. In the majority of these the macular lesion appeared merely haemorrhagic, but in a certain proportion there were seen the so-called "holes." Such macular disturbance, not at the point of contrecoup but more in the region of the equator round the line of impact, is difficult to explain, but may possibly be accounted for by the fact that the macula, being the most delicate part of the retina, is more liable to be injured by sudden increase of intraocular pressure than other parts of the retina.

Peripheral Traumatic Holes. Peripheral holes may certainly occur adjacent to the site of impact as in the case I have already cited, where the foreign body struck the outer side of the eye, and the hole in the retina was found in the outer periphery. This corresponds with our experience during the war of concussion injury of the eye, where on microscopic examination the retina was frequently found ruptured adjacent to the site of impact.

But peripheral holes also occur after trauma in the protected parts of the eye, where they could hardly be due to direct impact. It is possible that these ruptures of the retina correspond with the

ruptures of the sclerotic seen in civil practice following direct impact from slowly moving objects, and are due to the blow causing distension in the equator round the line of impact. The sclerotic containing elastic fibres is more resilient and does not rupture so readily, but the retina having no elastic fibres gives way before pressure. If this be the cause, however, it is surprising that these cases of peripheral rents, not occurring at the site of impact, are not more frequently associated with macular holes also.

Non-traumatic holes. Passing now to the non-traumatic holes, we find various theories put forward to account for their occurrence.

(1) We must certainly place cystic degeneration as a likely cause.

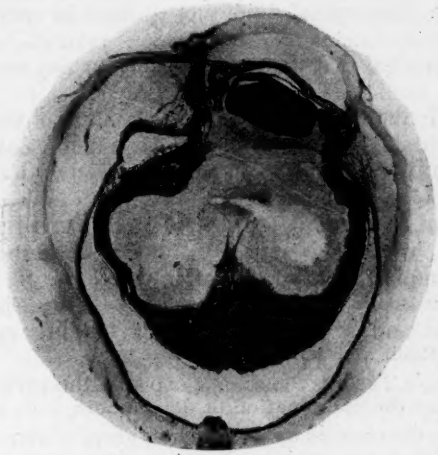


FIG. 1.

A large cyst or numerous small cysts having formed from whatever cause, gradual expansion of the cyst with consequent thinning of the walls may bring about rupture, or the latter may be determined by some slight trauma from without or traction of adherent vitreous from within.

I have recently seen a case of superior detachment in a myope in which there were several holes, over two of which retinal vessels coursed. In one hole an island of retina appeared to lie unattached, though there may have been some point of connection with the rest of the retina which was invisible.

I cannot offer a wholly satisfactory explanation of this, to me very unusual, case, but would suggest that it is probably due to rupture of cysts.

(2) *Raehlman's osmotic diffusion theory.* When an intact retina separates a highly albuminous exudate in the inter-retinal space from a watery vitreous, diffusion from the vitreous to the albuminous exudate might be expected to take place, which would increase the area of detachment. Raehlman suggests that by this diffusion of fluid the tension in the inter-retinal space may become so much greater than in the vitreous, that rupture of the retina takes place.

This theory of the formation of holes has several objections.

(i) Have we any evidence that osmosis takes place through the living and intact retina? Though theoretically it might take place, it appears very doubtful if much diffusion from within outwards actually occurs. For in long standing cases of detachment due to sarcoma of the choroid, where after fixation the vitreous remains clear and the inter-retinal fluid sets solid, it is obvious that the inter-retinal fluid remains more albuminous than the vitreous; one would have expected that if osmosis occurs to any marked degree, the two fluids would not remain so dissimilar.

(ii) Considering the extremely easy separation of the retina from its pigment epithelial layer, one would expect that should a difference of pressure on its two sides occur, it would merely lead to increase in the detachment. A difference of pressure on the two sides of the retina sufficient to cause rupture, could only arise if the retina were pegged down by previous retino-choroiditis, or if the detachment was nearly complete.

Therefore it seems probable that holes in the slighter degrees of detachment and in the absence of choroiditis can hardly be explained by Raehlman's theory.

(3) *Elschnig's Theory.* Elschnig explains the presence of holes in the retina by the combination of detachment with a pegging out of the retina to the choroid by pre-existing areas of retino-choroiditis. If the detaching force, whether traction from within or pressure from without, be sufficient to tear the retina, holes will be produced where it is pegged out. On the other hand when the retina is too tough to tear, it will remain adherent to the choroid in a funnel-shaped cord at these spots.

It is disappointing that in all the reported cases of holes due to pegging out of the retina by choroiditis, which I have come across, the accounts have been incomplete. Thus in Elschnig's first reported case there was a patch of retino-choroiditis seen ophthalmoscopically, and at a later examination a hole in the retina was found corresponding in position with the spot of retino-choroiditis, but the condition was not substantiated anatomically. In his second case there was a large hole in the retina and through it degenerated choroid was seen, but it was not proved that a piece of retina was torn out.

In Collins' two cases, patches of degenerated retina were found

attached to the choroid but the holes in the retina were not seen, though they must have existed in the contracted retina.

In spite of the reports being thus incomplete it is quite evident that Elschnig's theory is a true explanation of certain cases.

A somewhat rare but allied condition illustrates his theory, namely *total detachment of the retina.* (Fig. 1.) In such cases though the hole is not seen except on microscopic section, the retina being contracted up into a mass behind the lens, the disc is found with no retina attached, showing that the latter must have been completely torn away.

A different result will occur according to the nature of the detaching force, i.e., whether there is "a pushing from without" by fluid secreted into the inter-retinal space, or by possible osmosis; or "a dragging from within" by an adherent and contracting vitreous.

FIG. 2.

If the force is pushing from without, directly one hole occurs, equalisation of pressure on the two sides of the retina will be brought about and no further detachment or tearing will occur; that is to say only one hole can be produced by pushing from without. If, however, the detaching force is traction from within, then as many holes may be formed as there are points of attachment, provided the detachment is sufficiently extensive.

I examined the eye of a doctor who, when lowering his head to look down a microscope, ran a knife into his eye. There was a wound of the sclerotic at the limbus, the cornea was hazy with striate keratitis, the anterior chamber was deep, the iris muddy, the lens uninjured. The vitreous was shrunken to 1/6th of its normal size and there was haemorrhagic infiltration at the site of the wound, but it was in no way visibly attached to the retina. The retina was detached and in it were three circular punched out holes, one by itself and two together with the edges inverted. (Fig. 2.) Opposite

the holes were small whitish spots on the choroid, while in several other places the retina was attached to the choroid. Sections were cut at the site of the holes and also where the retina was adherent.

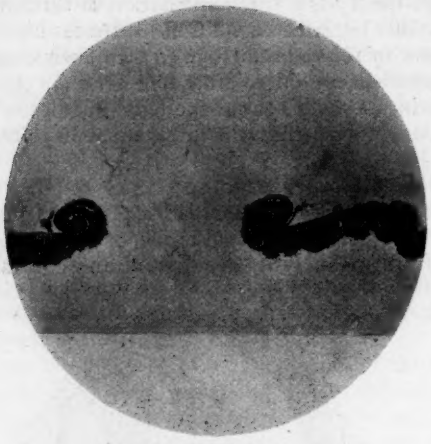


FIG. 3.

FIG. 4.

(Figs. 3, 4, 5.) In spite of serial sections being made I could find no trace of retina remaining adherent to the choroid opposite the holes; but there was some laminated new-formed fibrous tissue, which evidently gave rise to the white spots seen macroscopically.

These fibrous patches were rich in cells similar to those which were attaching the retina to the choroid at the points of adhesion. (Fig. 6.) Though in this case the holes were obviously associated with choroiditis I am unable to decide whether the retina was "pushed in" or "pulled in." The vitreous was demonstrably contracted, but between the shrinking vitreous and the retina

FIG 5.

FIG. 6.

there was a layer of clear limpid fluid; if therefore the retina was "pulled in," why were no points of adhesion of the vitreous to the retina discovered? While if it was "pushed in" how could the presence of three holes be accounted for?

The retina round one of the holes was much degenerated and cystic (Fig. 7). and therefore obviously very friable, so that very slight traction would be sufficient to cause a rent. It is possible the vitreous had previously been adherent and dragged on the retina, causing it to tear, while at a later date the points of adhesion to the retina had given way.

Though unable to explain the actual mechanism, this case is

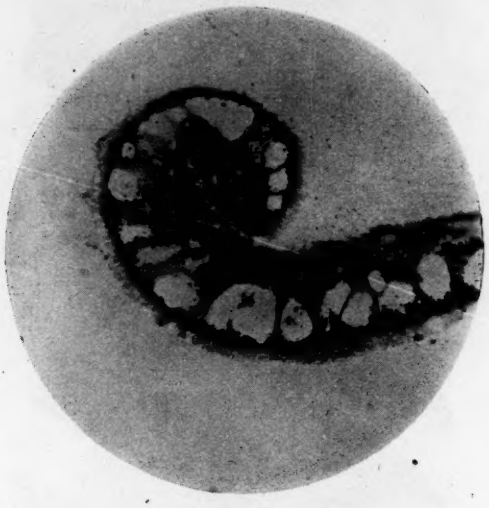


FIG. 7.

brought forward to illustrate the forms of holes associated with retino-choroiditis.

(4) *Leber and Nordenson's theory* of traction by fibrous bands in the vitreous. That macroscopic fibrous bands in the vitreous attached to the retina may cause folding, crinkling and detachment of this membrane is well known; we have all seen cases of retinitis proliferans ophthalmoscopically, giving rise to this appearance, and the following specimens of traumatic retinitis proliferans demonstrate them. (Figs. 8, 9.)

Such fibrous bands most frequently follow a haemorrhage from the retina into the vitreous from trauma, or are possibly—as Flemming suggests—associated with some toxic inflammatory condition; the vitreous thus becomes adherent to the retina at

the site of the haemorrhage, and, on its shrinking and becoming separated elsewhere from the retina, the latter is dragged on and becomes detached. (Fig. 10.)

In other cases attachment of the vitreous to the retina follows plastic cyclitis when fibrinous exudate is poured out into the vitreous which subsequently contracts. The following sections of a case

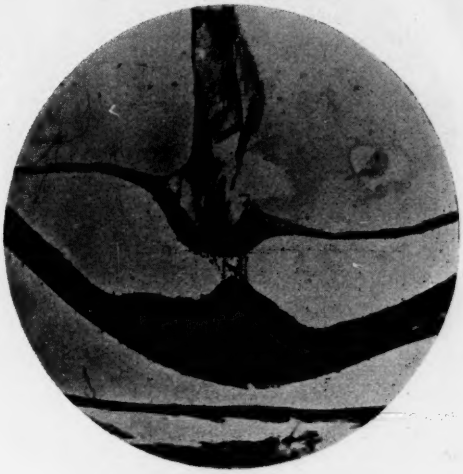


FIG. 8.

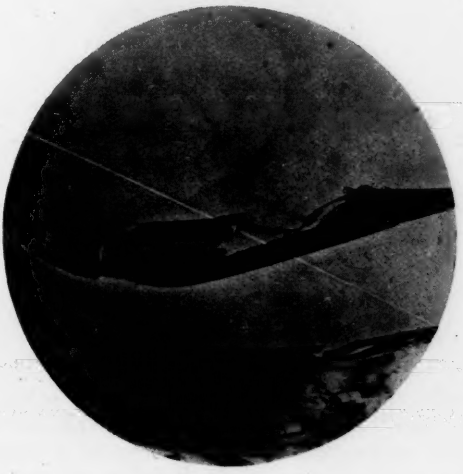


FIG. 9.

of plastic irido-cyclitis showing fibrinous exudate in the vitreous, detachment of the retina, and detachment of the choroid demonstrate the festooning of the retina between its points of adherence to the choroid caused by the contraction of the vitreous. (Figs. 11, 12.) If the adhesions, either of the vitreous to the retina, or of the retina

FIG. 10.

FIG. 11.

to the choroid had been stronger, it is easy to see that holes could have been formed, either according to Leber's or Elschnig's theories.

FIG. 12.

FIG. 13.

To show that a fibrous and shrinking vitreous adherent to the retina may cause holes apart from choroiditis, I may quote one of Lawford's cases, a woman aged 60, whose sight failed suddenly. Ophthalmoscopically no details were visible. There was only a red

fundus reflex downwards and down and out on focal illumination, and there was much floating vitreous opacity. A growth was suspected and the eye excised, and I had the good fortune to examine it. (Fig. 13.) The anterior part of the eye was normal, the vitreous was brownish and contracted to less than half its normal bulk and fibrous in consistency. Between the vitreous and the posterior part of the retina was a space filled with clear fluid, but from the ora serrata to near the region of the equator the vitreous was adherent to the retina. The retina was detached all round from the ora serrata to the disc, and the subretinal fluid was clear. At one spot near the ora, the retina was so much puckered and adherent to the vitreous that I suspected the presence of a foreign body. This was, however, excluded by X-ray examination. At one situation close to the junction of the retina and shrinking vitreous there was a hole in the retina 2 mm. in diameter. Its edges were inverted and from its anterior edge there projected a flap, which was firmly adherent to the vitreous. Could this flap have been reflected back into place, it would have turned the hole into a horse-shoe shaped rent. Careful search failed to reveal any sign of choroiditis or disturbance of the retinal or choroidal pigment opposite the hole or elsewhere. This case appears to be obviously one of a hole in the retina torn by an adherent and shrinking vitreous.

Sometimes we see with the ophthalmoscope horse-shoe shaped rents in which the central flap is turned inwards and has some thickened vitreous attached to it. Such a condition strongly suggests that an adhesion of vitreous has taken place, which by dragging on the retina has brought about a rent.

It is thus clear that macroscopic fibrous bands can cause both detachment and rents in the retina; but how are we to explain the numerous cases of holes met with clinically in which there is no history of trauma, no sign of choroiditis which might peg out the retina, and no marked opacity of the vitreous—merely a few webs or floating dust—but nothing definite in the way of bands? Are all these cases of burst cysts, or can there be tags of vitreous adherent to the retina, which are on the one hand sufficiently strong to cause traction and rupture of the retina, and on the other so fine as to be invisible with the ophthalmoscope? It seems to be a matter which needs further investigation, for as we shall see in dealing next with the clinical significance of holes, it is a matter of the greatest importance to decide whether the retina has been "pushed in" or "pulled in."

We may hope that possibly in the future, examination of the vitreous with the slit lamp may give us valuable information on the point.

III. Clinical Significance of Holes in the Retina

There is a very important fact, which Leber mentioned in his lectures at Heidelberg in 1882, but to which I think too little notice is given—namely that *sudden detachment of the retina cannot occur without a hole.*

In taking the history of cases with loss of sight in an eye, it is important but often difficult to distinguish between those in which defects in sight have existed for weeks or months but have been suddenly detected, from those where the loss of sight was actually sudden. The defect in an eye is so frequently only discovered on closing the other, and the individual is liable to think he has lost the sight suddenly, when in reality it has been defective for a long period, or possibly never has been good.

Sometimes, however, there is evidence in the history which distinguishes the two groups. A man may have noticed that one eye was uncomfortable or the sight impaired in some way, and have compared over some long period the sight in his two eyes, and then on a certain day—it may be, he can time the exact moment—the sight in one goes; such evidence is of great clinical significance especially in cases of detached retina.

With an intact eyeball and an intact retina, no detachment can take place unless there is a simultaneous and practically corresponding outpouring of fluid into the inter-retinal space on the one hand, and contraction of the vitreous on the other. Such pouring out of inter-retinal fluid and absorption of the vitreous cannot of course take place suddenly.

The onset of any detachment of the retina without a hole must therefore of necessity be gradual; whereas if there is a hole in the retina, transposition of fluid from the vitreous chamber through the hole into the inter-retinal space can readily take place, floating up the retina and causing a sudden loss of sight.

It would therefore appear that if it can be substantiated in any given case that the loss of sight from detachment was not merely suddenly detected, but actually occurred suddenly, we can definitely predict the presence of a hole, whether or not it can be seen by the ophthalmoscope.

A medical student was coming home late one night and was set upon by a lot of rough boys who knocked him down and hit him about the head and body. He did not notice that his eye specially had been hit, and being an intelligent and acute observer it is practically certain that he would have detected defect in the sight of either eye had any been present.

He, however, noticed nothing wrong with his eyes till eight days after, when, while walking up the outside steps of an omnibus, the sight suddenly failed in one eye. He was seen soon after at the

London Hospital, and found to have a large detachment of the retina in which there was a big rent.

I think the reasonable explanation of his case is that one of the many blows he received fell on the eye and caused a hole in the retina, through which eight days later, on some sudden movement of the globe the vitreous passed and floated up the retina.

Transposition would certainly take place more easily when the vitreous is fluid—or when "detachment of the vitreous" has taken place and fluid fills the space between the contracted vitreous and the retina. Whether transposition of the normal jelly-like vitreous could take place through a hole is doubtful. Collins suggests that the reason why some holes are not associated with detachment is that in these the vitreous has notdegenerated and therefore is not fluid and does not pass through the hole.

A second point of clinical importance is that, so far as I am aware, *no cases of detachment of the retina due to neoplasm, when examined pathologically, have been found associated with a hole in the retina.* This is supported, so far as my own experience goes, by the fact that in every case of neoplasm the inter-retinal fluid sets solid on fixation, which I think must be a proof that there is no hole in the retina.

If it could be definitely proved that holes in the retina do not occur with a neoplasm, we should have a very important clue in the differential diagnosis of causes of detachment—namely, that if we find a hole in a detached retina we can exclude neoplasm.

It is unnecessary to labour the importance of such a diagnostic point for we are constantly seeing cases of detached retina in which there are no positive signs of neoplasm, but where we wonder whether "behind the veil" there lurks a malignant growth. Thus there may be no new vessels seen in the detached area, no abnormal pigmentation of the retina, no brown reflex on focal illumination, no shadow on transillumination and no congestion or pigmentation of the sclerotic seen externally. There may be no history of a blow, no myopia, no sign of cyclitis, either keratitis punctata or vitreous opacities, and no retinitis or choroiditis and yet perhaps the position of the detachment is one unaccounted for by gravity. Any safe clue to diagnosis would be welcomed. The presence of a hole in the retina in such a case would, if we are correct in the above observation, give certain grounds for exclusion of a growth, and justification in leaving an otherwise suspicious eye unexcised.

To give an instance: a woman, aged about 50, came to the hospital complaining of failure of sight, which had come on gradually. She had noticed a curtain, as it were, drawn across her sight from the outer side. On examination a large detachment was found on the nasal side and also below. In this case there was no history

of blow, there was no myopia, no signs of cyclitis, retinitis or choroiditis, but there were some slight vitreous opacities. The detachment appeared quite "simple"; there were no new vessels or pigmentation of the retina, no sign of pigmentation or congestion of the sclerotic. The fact, however, that the detachment remained on the nasal side in a position unaccounted for by gravity, made me think there must be a growth on that side which was the cause of her trouble. I advised excision, but she refused to have the eye removed. It was not until a fortnight later that I found a large hole in the retina. I then felt justified in telling her that excision was unnecessary, and the subsequent history showed that this view, in her case at any rate, was correct. The eye gradually degenerated; it developed glaucoma, lost all perception of light and became painful. She then consented to have it removed. On pathological examination no growth was found.

I have recently seen another case of an elderly man who had a globular detachment limited to the upper part of the retina. There is no myopia, no history of a blow, no choroiditis or retinitis, no keratitis punctata, and only very slight vitreous opacity, but in the retina where it is detached there is a large rent, through which not only can one see the choroid but one has also the unusual opportunity of inspecting the posterior surface of the retina (Plate II). In this case, if there had not been a hole, one would have strongly suspected the presence of a growth.

To recapitulate the above remarks—I would suggest that (1) If we can detect a hole we can exclude growth, and (2) If we can substantiate a history of sudden loss of sight from detachment, even though a hole cannot be seen, we know that a hole must be present and we can with equal certainty exclude growth.

There is a third point of clinical importance which I should like to discuss, viz., ***the value of treatment of detachment of a retina in which there is a hole.***

Treatment of detached retina (irrespective of the presence of a hole) is admittedly most trying to the individual and uncertain in its results, yet this must not of course deter one from enterprise in suitable cases, for when treatment *is* successful, the boon to the patient is inestimable. But I think we need to exercise the greatest care in determining which cases are suitable and which are not, in order that a good procedure may not be undervalued by the profession, and the public may not have its confidence in the profession undermined by unsuccessful treatment.

We need every help to group our cases into those in which operation holds out the possibility of success and those in which no treatment can succeed.

If we divide our cases of detachment as far as possible by their aetiology, we recognise that the retina is either—

(1) *Dragged* in by an attached and shrinking vitreous, or by interstitial contraction as in retinitis striata.

(2) *Pushed* in by fluid poured out from the choroid into the inter-retinal space owing to some low form of inflammation.

or (3) It may be *floated in* by transposition of the vitreous through a retinal hole.

I. *Where no hole in the detachment can be detected,* I would suggest that—It is in the cases where we may reasonably suppose that the retina has been "pushed in" by fluid in the inter-retinal space—apart of course from cases of neoplasm—that we may hope for success in treatment.

Also in the cases in which we can find no cause for the separation of the retina, we may recommend treatment in the hope that a proportion may be benefited.

When however we can substantiate that the retina is being dragged in by vitreous bands or by cicatricial changes in its substance, no treatment at present devised is, as a rule, of any avail and should seldom be attempted: it is labour lost, a valueless strain is made on the patient and a needless postponement of adjustment to his limitations. Von Hippel's brilliant case, in which he divided a cicatricial band and the retina went back and useful vision was restored, is very exceptional, for a single definite band is of great rarity.

II. *Where a hole can be detected* in a detached retina, is treatment of any avail? So far as my experience goes, when there is a hole in the retina, the detachment is *usually* extensive and the vision correspondingly poor; there are, however, exceptions. Thus (i) macular holes, as mentioned above, are for some reason seldom associated with much detachment. Why transposition of the vitreous through such holes does not usually occur is not so far as I know yet explained: possibly it is the jelly like condition of the vitreous or possibly some macular holes are not real holes but only cysts. (ii) Certain peripheral holes in the retina may also occasionally occur with no detachment, as in the case I have already mentioned, or with a detachment which remains strictly localised. I have recently seen a case where there was a small strictly localised detachment in the retina below, following trauma in which there was a large rent. This has remained stationary for four months and the eye has retained perfect acuity of central vision—6/6ths and Jaeger 1—with a practically complete field, there being only a slight contraction above. It is probable that here the retina is bound down by choroiditis at the base of the detachment, for there is a definite disturbance of the retinal and choroidal pigmentation in this region, and that it is for this reason the detachment remains stationary.

In such cases clearly no treatment is indicated.

In the majority of cases of peripheral holes, however, detachment is extensive.

Leber considered that a hole in the retina was essential to its re-attachment, since through it the fluid in the inter-retinal space can pass back into the vitreous chamber and the retina become reseated. It is difficult to understand, however, if reseating of the retina takes place with rest alone, what is to maintain it there, and prevent its being floated up again directly the eye is moved, unless there is plastic choroiditis present to hold it in place, a very unlikely concomitant.

In the report of the Committee appointed by the Ophthalmological Society of the United Kingdom to collect data with regard to the cases of cure after detachment of the retina, three cases were mentioned in which there was a hole where cure was effected without operation; but in no case with hole was success obtained by means of operation.

Good results have been obtained by many surgeons, who after draining the inter-retinal space have punctured an intact retina; but this does not appear to me to be a true indication that cases of detachment with holes are responsive to treatment. There is surely a considerable difference between draining the inter-retinal space with subsequent puncturing of an intact retina and the attempt to drain the inter-retinal space where there already exists a retinal tear. In the former we may expect the inter-retinal space to be practically emptied of fluid, and the retina approaching the pigment epithelium before the puncture in the retina is made; the wound of the retina too might help in the glueing of the retina into position. Whereas if we attempt to drain an inter-retinal space with an already existing retinal tear, we should be draining the vitreous through the hole almost as much as draining the inter-retinal space.

I have heard of one case in which there was a hole, where the retina went back after operation, but the improvement was only temporary; the detachment subsequently reappeared.

Personally I have seen no case in which there was a hole improve either spontaneously or after treatment, and it is my opinion that with our present available methods such cases of detachment are practically hopeless. The experience of one man is however of little value, and in order to answer the question whether, with our present methods, treatment of cases of detachment with holes should be undertaken or not—we require—

(1). Increased knowledge of the causation of holes,

(2). Improved clinical methods of detecting whether the retina is being pushed in by fluid from without, or dragged in by adherent and contracting vitreous, and

(3). A larger number of reports of the practical experience of

surgeons in the treatment of these cases, classified according to their cause.

Not until we have a fuller knowledge of their aetiology can we reasonably hope either to advise rationally or to devise a more successful procedure in the treatment of these difficult cases.

THE RELATION OF EXOPHORIA IN EARLY PRESBYOPIA TO REFRACTIVE ERRORS*

BY

Dr. LUTHER C. PETER

PHILADELPHIA, PA.

ROUTINE studies in any particular line of cases are rewarded by the uncovering of many unsuspected, but none the less prevalent, conditions. The practice of routine muscle studies in refraction is no exception to the rule. One of the outstanding facts in this special line of investigation is the gradual increase in the number of exophorias encountered from the age of thirty to that of forty-five or fifty. Two distinct types of cases are to be found in this period. First are those which simply represent a carry-over, as it were, of an aggravated condition which has its inception in early adult life and increases with the added strain of waning accommodation in the presbyopic period. These fortunately make up the smaller group. From an aetiological standpoint, as well as from the standpoint of possibilities in treatment, they furnish a fascinating field for study. The second or larger group are those which one may appropriately call "accommodative exophorias"— exophorias which have as their basis failing accommodation. This group includes so many cases which need our careful consideration, that they alone will be discussed.

The underlying factor is failing accommodation. A summary, however, of case records seems to point to one fact especially significant, a majority of these patients have never worn glasses or have been improperly glassed until the age of thirty-five or later. If glasses are properly prescribed in early adult life, and again as the presbyopic age creeps on, this type of exophoria is comparatively infrequent in occurrence and is easily managed. In the writer's experience, those, who have most symptoms, have the greatest degrees of exophoria, and are the most difficult to handle, are the hypermetropes who have failed to wear suitable glasses until the early presbyopic period.

*Read before the Ophthalmic Section of the College of Physicians of Philadelphia, October, 1923.

Two or three dioptres of hyperopia with low grades of astigmatism may cause little trouble before the patient is 37 years of age. At this period, however, even a moderate degree of hyperopia begins to inconvenience the patient in close work and he looks for help. Still unwilling to wear glasses constantly or possibly uncomfortable with a full correction because of an overacting ciliary muscle, a full correction is usually prescribed for near work and distance glasses are not worn. The consequence of this method of handling or indulging the patient is a disturbance of the relation between accommodation and convergence.

There is a temporary period in most cases, even if properly glassed with a far and near correction, when exophoria may appear as the result of the sudden relaxation in accommodation incident to the wearing of the near correction. In the average individual this readily adjusts itself or may only be present in fatigue. In the patients, however, just referred to, it becomes necessary at all times to utilize an excess of their reserve accommodation for distance alone. The normal relation of the metre angle of convergence to the dioptre of accommodation is not maintained at infinity and is not observed at the working near point. Convergence is repressed in the interest of single binocular vision.

It is generally assumed that convergence excess is the rule when an excess call is made upon the accommodation for clear distant vision, and on this belief is based the accommodative theory of the aetiology of convergent squint. Donders, however, has clearly shown (Accommodation and Refraction of the Eye, 1864, pp. 110-111), that each may work independently of the other in the interests of clear binocular vision. Operating over a period of many years, a new relation, therefore, may be established between accommodation and convergence for a given patient with two or more dioptres of uncorrected hypermetropia.

At this period of disturbance, the optician prescribes rest glasses; the careless oculist prescribes without drops; or because the patient objects to glasses for constant use, or the ciliary muscle rebels, a two or three dioptre reading glass is prescribed and the distant vision is uncorrected. Convergence, which is already repressed, fails to respond normally, with a total relaxation of accommodation, and exophoria, often of ten to fifteen degrees, develops.

A fair statement of the aetiological factors must necessarily include a small group of patients properly glassed throughout, who, temporarily, or when fatigued, or when somewhat below normal in health, may show moderate degrees of exophoria for the near point, especially when wearing their first presbyopic glasses. It is a form of exophoria which responds readily to proper therapeutic measures.

The symptoms complained of by these patients, aside from the difficulty of seeing clearly at the near point, are headache, usually

supraorbital and temporal, but often occipital, after doing close work; drowsiness on the slightest attempt at reading; redness of lids, vague gastro-intestinal disturbances, and nervous conditions usually attributed to other causes. Reading, which to the average man becomes more and more a habit and a source of pleasure and occupation as the years multiply, becomes intolerable. To the man whose work requires close application at the desk work becomes a burden. These patients are apt to go the rounds in an effort to obtain relief. Some are successful; others accept what they believe to be the inevitable and shun close work as much as possible, and may even change their occupations.

What can be done to correct the condition? The answer is not a simple one.

The first step is careful refraction, with a cycloplegic in all cases in which it can be safely used. To discuss the cycloplegic best suited would take us too far afield. Unfortunately, homatropin is now generally used as the drug for this period. There are cases, however, uncorrected throughout life, in which homatropin will be found to be inadequate to control properly an unequally developed ciliary muscle. In these cases, scopolamin should be used without stint for the double purpose of properly controlling the ciliary muscle during the examination, and in order to keep the muscle relaxed for a longer period than is our custom.

If exophoria is marked at this early period, a full distance correction, even if tolerated, should not be given at once. In most instances this advice is unnecessary, as few will accept two or three dioptres of correction immediately. Two pairs of glasses should be prescribed—a full correction for close work and a weaker correction for constant use. In many instances, the weaker correction can be discarded in a month or two, and the full correction will be accepted. In other instances, a second or a third change may be necessary before the full correction will be accepted. The wearing of a full correction will be determined largely by the patient's tolerance and by the results obtained by prismatic and stereoscopic exercises.

Some years ago, when inspired by the excellent work and teaching of the late Wendell Reber, the writer began to use prism exercises; either because the cases were poorly selected or the exercises were improperly conducted, the results obtained were rather discouraging. Necessity, however, is the mother of invention, and the number of these cases which make up an average clientele compelled more assiduous study with gratifying results.

The cases fall into two groups. Those most suitable for prism exercises alone usually manifest exophoria for distance and a greater degree of exophoria for the near point. Adduction is below normal. Those in which stereoscopic exercises, combined with prism exercises,

are best adapted have an esophoria for distance and exophoria for the near point. In the latter group both adduction and abduction may be above normal but improperly balanced.

The first object of prism exercises at six metres is to restore a normal relation between the dioptre of accommodation and the metre angle of convergence which has become viciously disturbed by the necessity of using too much accommodative effort at infinity. This is especially imperative in those individuals who are able to obtain normal visual acuity at infinity in spite of the presence of three and even four dioptres of hypermetropia. It is not easy to accomplish this, especially in patients who have gone uncorrected until they reach the age of thirty-five. Much time must be spent, and patient and doctor should have considerable patience, therefore, when treatment is instituted. A normal balance can be restored in most cases, if the technique is good and the exercises are continued sufficiently long.

The technique is so important that a brief enumeration of the important steps should not be amiss.

1. All exercises should be conducted at six metres with the light at proper level—not above the patient's eyes.
2. A full correction should be worn during the exercises.
3. Prisms should not be tilted up or down, but with bases out held perfectly level before the patient's eyes. It may be impossible to fuse if the vertical displacement of the light is even one degree.
4. The increase in prism strength should be very gradual at first, two or three prism dioptres at a time, until the patient has caught the knack of fusing.
5. *The patient must see the two images fuse into one.* One of the causes of failure is due to non-observance of this rule. Patients will return with a report of being able to practise with 70° and even 90° prisms at home, when actual experiment in the office may show a possibility of but 15 prism dioptres. The second light deviates so far to the right or left that they fail to recognize its presence.
6. Minute instruction must be given in the office, two or three times. The actual work is then conducted at home daily for a week when the patient should return and have any errors in technique corrected. Return visits after the first week should be made in two weeks and later once monthly.
7. Home practice should be conducted every day for 15-minute periods until satisfactory results are obtained.
8. If several months of treatment bring about satisfactory results, two 20° and two 15° prisms should be purchased by the patient and a few moments of practice should be indulged in at intervals.

The degree of exophoria which may be overcome by this method averages from two to seven degrees at infinity with a corresponding

seven to twelve for the near point. Long before the Maddox rod shows a normal or nearly normal balance the patient will report relief from the symptoms which led him to consult the oculist. Those who have high degrees of exophoria will also report comfort in close work in spite of a residual degree of exophoria, amounting to as much as seven or eight degrees for the near point as shown by the Maddox rod test. The explanation of the relief from asthenopic symptoms, even though the Maddox rod registers so much exophoria for the near point, lies in the fact that the nearness of the images and their similarity enable fusion to accomplish its work. Failure cannot be entirely determined by the Maddox rod. The patient's comfort must be the deciding factor in high and sometimes even in low degrees.

Improvement in abducting and adducting power and their relation are important, but, after all, their relation is disturbed primarily by the vicious interference referred to early in the discussion. The first object, therefore, is to correct, not an inherent duction weakness, but an unbalanced relation which has been brought about by failure to correct hyperopic defects early in life.

The role of the stereoscope is also important. It is too intricate, however, to discuss in detail in this communication. It is sufficient to say that it is a most excellent instrument with which to supplement prism exercises as a stabilizer, especially when both adduction and abduction are well developed but are not well balanced. It is of greatest help in those cases in which there is an esophoria for distance and an exophoria for the near point. Its proper technique is as intricate as prism exercises and deserves a separate communication for its proper presentation.

The writer cannot close this discussion without a final word to those who refract without routine muscle studies and to those who have tried prism exercises and have failed to obtain satisfactory results. Those who refract without routine muscle studies, whether in private practice or in the hospital clinic, are not doing justice either to themselves or to their patients. In private practice there can be no excuse for this omission. In hospital work there should be no excuse. The taking of muscle balance does not require an expensive equipment, nor does it require much time. In hospital work, if muscle defects are found and their importance is explained to the patient, most patients will be willing to find the means for private office training. Or, failing in this, the responsibility rests with them. Prism training is too time-consuming for dispensary application.

Those, on the other hand, who have practised prism training and have failed to obtain results should try again, with a change in technique. The results which the writer of this paper has obtained are being duplicated by many. They are offered only after having been thoroughly tried and not found wanting.

THE DIFFERENTIAL DIAGNOSIS BETWEEN PARINAUD'S CONJUNCTIVITIS AND CONJUNCTIVITIS NECROTICANS INFECTIOSA

BY

PROF. C. PASCHEFF

SOFIA

IN spite of numerous recent publications, more especially in German Ophthalmological literature, on the subject of Parinaud's conjunctivitis, the question of the nature of the disease remains obscure. Thus Bayer and Herrenschwand[1] in two cases of an unusual type come to the conclusion that they were cases of Parinaud's conjunctivitis and, in spite of the fact that no histological examination was made, classed them as pseudo-tuberculosis. Others consider the disease to be a manifestation of tuberculosis; Verhoeff[8] proposes to call the disease leptothrix conjunctivae, and so on. In France and in Germany opinion is generally against the theory that the disease is due to tubercle. All this shows that there is a certain similarity in the symptoms of all these cases, but that there are different causes and also differences in the histological appearances which have unfortunately not always been investigated. If in all these cases histological and bacteriological examinations had been made we should have had a more definite idea of the nature of the disease. The only undoubted similarity of all these published cases can be reduced to a few clinical symptoms which I propose to term "Parinaud's syndrome." These are the presence of a unilateral conjunctival inflammation accompanied by suppuration in the preauricular and submaxillary glands and a rise of temperature. Thus, just as in Mickulicz's disease, we have to deal not with a definite disease but with a syndrome of symptoms due to different causes.

In 1915 I separated from the chaos of so-called Parinaud's disease a type of conjunctivitis with definite symptomatology, evolution, pathological anatomy and aetiology which I called "Conjunctivitis Necroticans Infectiosa." In my researches on this disease I discovered one definite criterion that served to distinguish it from other cases of Parinaud's disease. This was the effect of inoculation on guinea pigs and rabbits which caused the death of these animals with characteristic white spots in the spleen.

As this criterion is the basis for differentiating the disease I have described from that described by Parinaud and his followers, I propose to give a brief comparative resumé of my results and those of Parinaud in the hope of creating a better base for future research work in this connection.

I. Symptomatology and Evolution

Parinaud's conjunctivitis. (Three cases observed by Parinaud.)

A.—*General symptoms.*

1. Unilateral swelling of the parotid with reddening of the skin followed by either suppuration or resolution.

2. Rise of temperature.

B.—*Local ocular symptoms.*

The eyelids of the affected eye are swollen. Big granulations or red vegetations are found especially in the fornices and bulbar conjunctiva. They are translucent and pressed closely together (see Fig. 241 in Terson's "Ophtalmologie du Medicin Practicien," 1921). In places, especially over the sclera, yellow granulations, much smaller and closely pressed together are found. On the upper lid the vegetations show on their surface small polished areas but no regular ulceration. The secretion is mucous with some fibrinous deposit in the lower fornix. One of the vegetations reaches on to the lid margin. In another of Parinaud's cases the vegetations were of similar character but even more extensive.

Conjunctivitis necroticans infectiosa. (Three cases observed by me and one by Dr. Gunther.)

A.—*General symptoms.*

1. Unilateral swelling of the preauricular, parotid and submaxillary on the affected side, followed by suppuration in three cases and resolution in one.

2. Rise of temperature.

B.—*Local ocular symptoms.*

The eyelids of the affected eye are red and swollen. In the inferior conjunctival fornix there are numerous small white spots, the size of a millet seed, not sharply defined but merging into the surrounding red conjunctiva. Some are slightly elevated others excavated and ulcerated like phlyctenules. On the surface of the elevated spots there is a fine vascular network. These points and spots are to be found in the upper fornix without any vegetations (Fig. 1). They do not resemble ordinary follicles or trachoma granules, but, as Gunther remarks about his case, give rather the impression of superficial necrotic foci such as I have previously described. Another characteristic of these spots is that, after ulceration, they clean and cicatrize while similar fresh spots appear in other parts of the conjunctiva. In only one case I observed in the lower fornix a deep round yellow focus forming a small abscess (Fig. 4).

Fig. 1.

Fig. 2.

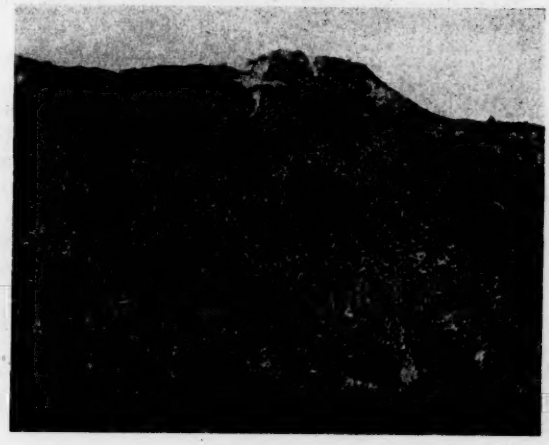


Fig. 3.

Evolution.

In Parinaud's first case two months after continued cauterization with silver nitrate the granulations were still present. In a second case they disappeared in two months.

II.—*Pathological anatomy.*

Parinaud gives none. According to Morax the epithelium is normal without any breach of surface. The conjunctiva is thickened and infiltrated by lymphocytes with some macrophages similar to those described by Villard. There are no giant cells. In some cases distinct follicles surrounded by thickened conjunctiva are formed.

The necrosis described by Derby and Verhoeff differs from that found in my cases in the absence of polynuclear leucocytes and in the nature of the cause.

Reiss draws attention to the presence of plasma cells and considers that the necrosis described by the last two authors is accidental and has no connection with Parinaud's disease.

Evolution.

The inflammation is acute. The spots disappear in from two to three weeks. The glandular affection may last from two to three months. The evolution of the white spots passes through three stages:—

1. Period of necrosis during which their formation takes place.

2. Period of ulceration during which they ulcerate and become clean (Fig. 3).

3. Period of repair and cicatrization.

II.—*Pathological anatomy.*

The white spots in the conjunctiva are the result of a dense infiltration in the sub-conjunctival tissue. This forms a focus of exudation which consists of—

1. Polynuclear leucocytes.
2. Tissue cells in active proliferation.
3. A peripheral zone of lymphocytes.

In the foci necrosis rapidly develops. If the infiltration is superficial it ulcerates and cicatrizes quickly, if deep it may develop into an abscess (Fig. 4).

The presence of many polynuclear leucocytes indicates the suppurating tendency of the disease with the rapidly formed central necrosis.

III.—*Experimental inoculations.*

Parinaud[5], Morax[4], Chaillous[2] and Gourfein[3] failed to get any positive results. The last author made numerous experiments on rabbits and guinea-pigs with material from a case in which granulations were present and came to the remarkable conclusion that Parinaud's disease cannot be transferred to animals. His case was cured in fifteen days but the adenopathy lasted six months without any suppuration.

III.—*Experimental inoculations.*

In all my cases, and also in that of Dr. Gunther, I succeeded in inoculating guinea-pigs subcutaneously and rabbits in the anterior chamber with excised portions of the affected conjunctiva. The guinea-pigs died in seven to eight days, the rabbits a little later. In both animals I always found in the spleen numerous small white necrotic points rich in polynuclear leucocytes and showing very early coagulation necrosis. I was thus able to reproduce the conjunctival white spots in the spleen of these animals and so establish a base for further research (Fig. 2).

Fig. 4.

IV.—*Aetiology.*

Bacteriological results of Parinaud, Gourfein, Chaillous and Morax were all negative.

IV.—*Aetiology.*

I succeeded in isolating in all the cases an organism to which I gave the name of "Micrococco bacillus polymorphicus necroticans." The morphological and biological characteristics of this cocco-bacillus are as follows (these results have been confirmed in Prof. Axenfeld's laboratory) :—

Size between 0·25 and 2 μ in culture. Shape usually that of a small coccus but sometimes that of a bacillus. It possesses neither cilia nor capsule and is Gram negative. It is both aerobic and anaerobic and does not grow at ordinary room temperatures. It does not liquefy Loeffler's serum or gelatine and forms no crust. It causes turbidity in bouillon, coagulates milk and gives an acid reaction. It does not form spores. It grows on the surface of agar without any colour and forms no gas; it is non-motile. Cultures must be renewed every seven to eight days.

Bordet and Gengou is positive with antigen 0·02. Agglutination with the patient's serum 1/2000. Experimentally, the disease could be produced in guinea-pigs exactly as with the affected conjunctiva and was fatal to them in the same time with the same post-mortem appearances in the spleen.

Since with these data in hand I was able to define a new kind of conjunctivitis with the syndrome of Parinaud. The opinion that Parinaud's conjunctivitis is not a definite disease but only a

syndrome of symptoms seems to be making its way in France.* Parinaud failed to describe fully a definite disease with its pathological anatomy and bacteriology and his followers have not been more fortunate but have only added to the obscurity. While some have described the clinical picture in very various terms, others have dealt with the histology and still others with the bacteriology. In order to know the true picture of the disease it is necessary that all these various observations should be correlated so that the clinical picture, pathological anatomy and bacteriology should all be defined. This has been done in the case of "conjunctivitis necroticans infectiosa" and I, therefore, propose to give it the first place under the heading of Parinaud's syndrome. The future will tell us what its relation to pseudo-tuberculosis may be.

The histological changes in my cases may be expressed in a few words as consisting in the formation of small abscesses with a suddenly formed coagulating infectious necrosis. Such appearances according to Schneider of Heidelberg have not previously been observed in man, nor has pseudo-tuberculosis. Pseudo-tuberculosis, as seen by me in the guinea-pig in Prof. Kossel's laboratory in Heidelberg is quite different from the appearances I produced. Bayer and Herrenschwand who consider that Parinaud's disease is a form of pseudo-tuberculosis give no histological evidence for their assertion as they have not examined the infected human conjunctiva. The pictures and results obtained by them in the guinea-pig are similar to mine that were published previous to their observations. After publishing his first case, Herrenschwand informed me that he had seen a case very similar to the one published by me and asked me to let him have all my publications on the subject. He subsequently changed his opinion as he observed in a second case (Bayer's) that follicles were present. These, however, do not show up in his picture (Fig. 6) and were not submitted to any histological examination. A histological examination might possibly have shown that his trachoma-like granulations were in reality small abscesses similar to that described in my third case (Fig. 3). Those interested in the subject would do well to compare Bayer and Herrenschwand's figure 6 with figure 241 in Terson's "Ophtalmologie du Medicin Practicien" in order to understand the views held as to Parinaud's disease in France and Austria.

It is, therefore, quite clear that "Conjunctivitis Necroticans Infectiosa" does not appear in the inflamed conjunctiva under the

*Beavieux in his work on Parinaud's conjunctivitis[10], says, "Pascheff in his paper on 'conjunctivitis necroticans infectiosa' (*Arch. d'Ophtal.*, Feb., 1921), considers this disease to be quite distinct from that described by Parinaud. All these different infections characterised by a general and glandular affection must be classed together under the title of 'Parinaud's syndrome,' but due to various causes. In spite of the fact that my bacteriological researches proved negative, I consider that my cases are to be closely compared with those published by the Bulgarian professor."

form of vegetations but of abscesses with central necrosis which, when on the surface, appear as white spots and ulcers (Fig. 3) but, when situated more deeply, appear as yellow grains. Further we are able readily to infect guinea pigs with the matter derived from these abscesses and white spots. The infected animals die quickly in from seven to eight days with characteristic white spots in the spleen. The microbacillus necroticans infectiosa was isolated in Dr. Angueloff's laboratory from these abscesses and with it we were able to reproduce the disease. This supplies the necessary test for the disease which is the characteristic infection of the guinea-pig. In spite of the simple nature of the test other observers (Chaillous, Morax, Gourfein, Reiss and others) have failed to obtain positive results in cases considered to be Parinaud's disease. It is on this account that I consider the experimental and histological evidence to be of much more value than any clinical appearance.

As in other granulomatous diseases tuberculosis, pseudo-tuberculosis, and actinomycosis, conjunctivitis necroticans infectiosa is a focal inflammation. It is an acute suppurative necrosing inflammation which rapidly kills inoculated guinea-pigs and rabbits forming in their spleens similar foci of exudation.

To confirm the diagnosis it is not sufficient to observe the white spots and yellow grains described above; histological and experimental investigation is also necessary. Others and I myself have found the white spots present in cases which gave no result on the guinea-pig. For inoculation purposes it is advisable to use the white spots mainly as these contain the more active organism.

In conclusion I consider that I am justified in classing these cases as a definite disease under the name of "Conjunctivitis Necroticans Infectiosa" and separating them from the general type of case known as Parinaud's disease.

BIBLIOGRAPHY

1. **Bayer and Herrenschwand.**—*Arch. f. Ophthal.*, p. 342, 1919.
2. **Chaillous.**—*Annal. d'Ocul.*, p. 1, 1907; *Soc. franç. d'Ophtal.*, p. 266, 1905.
3. **Gourfein.**—*Rev. méd. de la Suisse romande*, Feb. 20, 1907.
4. **Morax.**—*Encycl. franç. d'Ophtal.*, Vol. V, p. 774.
5. **Parinaud.**—*Soc. d'Ophtal. de Paris*, 1889.
6. **Pascheff.**—*Klin. Monatsbl. f. Augenheilk.*, 1916 and 1917; *Arch. d'Ophtal.*, 1921.
7. **Reiss.**—*Arch. f. Ophthal.*, Vol. LXIII, p. 96.
8. **Verhoeff and Derby.**—*Klin. Monatsbl. f. Augenheilk.*, p. 705, 1905.
9. **Schöltz.**—*Arch. f. Augenheilk.*, p. 705, 1905.
10. **Beauvieux.**—*Gaz. hebd. de Sci. méd. de Bordeaux*, March, 1921.

ABSTRACTS

I.—GLIOMA RETINAE

(1) **O'Connor, Dennis F. (Worcester, Mass.).—Glioma retinae et atrophia bulbi.** *Arch. of Ophthal.*, Vol. XLVI, No. 4, 1917.

(1) **O'Connor** believes that in his case the glioma was the primary condition; that the tumour after destroying the retina became almost completely necrotic; and that the necrotic areas became replaced by connective tissue in which the tumour cells again grew; the cell masses thus formed then underwent necrosis and calcification, probably from the lack of sufficient blood supply. He reviews the hereditary, prenatal, and congenital aspects of these cases, and discusses the available literature. Finally a section is devoted to the differential diagnosis. R. H. ELLIOT.

(2) **Rietz, E.—Glioma of the optic nerve.** *Hygiea*, Vol. LXXX, p. 344, 1918.

(2) **E. Rietz** remarks that time was when tumours of the optic nerve were believed to originate in its pial sheath, and were described as endothelioma, on the one hand, or as myxoma or fibrosarcoma, on the other. His own patient (Professor Dalén's clinic) was a girl, 3½ years of age. At the end of December, 1916, her mother noted that the child's right eye protruded somewhat, and later squinted inwards; a month later a watch could be seen a metre off with the left eye covered. Seen on February 17, 1917, the girl showed protrusion of the right eye forwards, inwards and downwards, with unimpaired mobility, loss of vision, enlarged pupil inactive to light but preserving the consensual reaction, iris of normal appearance, refracting media normal. The fundus exhibited a reddened prominent disc with radial striation and obscure margin; the veins were dilated and tortuous. The left eye was normal. Nasal examination showed nothing abnormal in the ethmoid region. Examination with the X-rays showed that the right lamina papyracea had less sharp angles than the left, and slight thickening in the ethmoidal region. Retrobulbar tumour was diagnosed and the right eye was removed. The optic nerve was club-shaped, hard, and even; no adhesions were present, and no orbital tumour could be felt. The optic nerve was cut as far back as possible, but its end showed evidences of the tumour, so the removal of the growth was incomplete. Five months later no signs of recurrence had occurred. Examination of the optic nerve after removal showed a spindle-shaped intrapial new growth, grey-white in colour, longitudinally striated; the arachnoid and pia were loosened and not

adherent, and there was no evidence that the tumour had broken through into the intervaginal space. The arachnoid membrane showed definite hyperplasia of the endothelium and connective tissue bundles. The eyeball was oedematous round the papilla, but not invaded by the tumour. The tissue of the tumour itself occupied the dilated interseptal spaces, which were trebled in diameter at the point of maximum enlargement, normal at the lamina cribrosa. The pial septa were thinned out in the tumour and vascluar. The tumour tissue was poor in cells, and showed the specific staining reactions of neuroglia, staining yellow with van Gieson's and red with Mallory's stain, blue by the aniline blue method. It consisted of long tortuous corkscrew fibrils in a network, staining red, while the connective tissue of the pial septa stained blue (staining method not stated). Staining for mucin gave a negative result. Neuroglia cells mainly of two types were seen, and a few nerve fibres and myelin sheaths; no necrosis was observed. Rietz says that two other cases of tumour of the optic nerve in the pathological collection of his clinic showed similar pictures; resembling gliomas of the brain rather than gliomas of the retina in their nature and course.

A. JEX-BLAKE.

(3) **Van Duyse, D. (Gand).—Homeotypical glioma of the optic nerve. (Gliome Homéotypique du Nerf Optique).** *Arch. d'Ophtal.*, December, 1922.

(3) In this article **van Duyse** describes very fully with numerous illustrations, the histological characteristics of a tumour of the optic nerve removed from a boy of 15, and designates the growth homeotypical glioma. Those interested should read the author's description which would lose much of its value if published in abstract. The author's concluding statement is that homeotypical glioma of the optic nerve has a glioblastic origin and is composed of differentiated neuroglial elements; it is a tumour with benign characteristics and is to be distinguished from heterotypical glioma —glio-sarcoma of some writers—which is essentially malignant.

In the introductory part of his paper van Duyse refers to the question of a non-malignant form of glioma of the retina to which attention has been directed by more than one writer. He says: "In an earlier communication (*Arch. d'Ophtal.*, March, 1922), I have described a homeotypical glioma which arose in the hyperplasic and colobomatous retina of a rudimentary eye with a cystic development behind the upper eyelid. I sought to establish the view of a form of glioma lacking the attribute of pernicious development associated with glioma of the retina, which has become an axiom in our clinics. The tumour was removed from an infant of eight weeks; 18 months later the child was in perfect health. I have since ascertained that

Sourdille (*Arch. d'Ophtal.*, 1904, p. 87), and Asceunce (*Annal. d'Ocul., 1905, p. 133*), have expressed similar views in reference to intraocular glioma of benign character. Without adhering to the views on histogenesis of the first-named writer, or the conclusions published by the second, I think that the occurrence of retinal tumours composed of differentiated elements, homeotypical 'adult' as Sourdille puts it, should be accepted in contradistinction to the more frequent, better known tumours composed of undifferentiated heterotypical 'embryonic' elements, which are metastatic and of evil reputation. Apart from the danger due to their situation this type of glioma (homeotypical) would be grouped among the benign tumours clinically. If this proposition were established in reference to the retina it would be equally applicable to the optic nerve."

To explain the difference between the two types of glioma Sourdille suggests that if the proliferation of the neuroglia is of the "adult" form a benign tumour results, while proliferation of the "embryonic" type produces a malignant growth. Histological examination alone will establish the diagnosis and the prognosis. Homeotypical glioma is benign in the sense that glioma of the brain and spinal cord is benign, i.e., a tumour of slow development which only exceptionally involves adjoining structures and is not metastatic.

J. B. LAWFORD.

(4) **Gourfein-Welt, Madame L. (Geneva).—A new aetiology of pseudo-glioma: sub- and pre-retinal haemorrhage in the course of whooping cough. (Nouvelle étiologie du pseudo-gliome: hémorrhagie sous et prérétinienne au cours de la coquelouche.)** *Rev. Gén. d'Ophtal.*, February, 1923.

(4) **Madame Gourfein-Welt's** case is of considerable interest and importance. A girl of five years who had had whooping cough in July and August came to the clinic in November. Except for the condition of the left eye she was in perfect health. The right eye was normal and emmetropic. The left eye showed external strabismus: otherwise its appendages, movements and tension were normal. There was no photophobia, ciliary injection or opacity of the cornea, anterior chamber or lens. The pupil was moderately dilated and did not react to light. Behind the lens was a greyish white mass with several vessels upon it. For safety's sake the case was treated as one of glioma retinae and the eye was excised. On pathological examination the condition was found to be quite otherwise. There were no evidences of glioma. There was a funnel shaped detachment of the retina with, in front of its post-lenticular portion, a mass of organised tissue of haemorrhagic origin, while, between the detached retina and the choroid was a greyish red mass which, from its microscopical composition, represented the remains

of a large sub-retinal haemorrhage. There was no ascertainable cause of the haemorrhages other than the whooping cough. The author occupies a good deal of space with description of the microscopic appearances, but the above notes will indicate the conditions found. Three drawings accompany the article.

ERNEST THOMSON.

II.—DISLOCATION OF LENS

(1) **Hegner (Jena).—Contribution to the prognosis of dislocation of the lens.** (**Beitrag zur Prognose der Linsenluxation.**) *Klin. Monatsbl. f. Augenheilk.*, Sept.-Oct., 1915.

(1) **Hegner** has collected 65 cases of dislocation of the lens in which he was able to follow up the after history; 70 per cent. of the patients were over 45 years old, a feature attributable to the changes which occur in the suspensory ligament with advancing years. For purposes of comparison five groups are recognised: subluxation, total dislocation into the vitreous, dislocation into the anterior chamber, congenital and spontaneous dislocation. In connection with subluxation secondary glaucoma is the greatest danger. It occurred in 20 out of 24 of Hegner's cases. The glaucoma may be acute or chronic, miotics and glaucoma operations are useless and extraction is dangerous. In eight extractions four eyes were lost and in only one was good vision obtained. The reason for such bad results is that changes in the vitreous and in the suspensory ligament are always present. In rare cases, however, spontaneous reposition may occur. Partial dislocations only rarely become total; this occurred in only one of 24 cases which were under observation for a considerable time.

Eleven cases of total traumatic dislocation were followed up. Of these only two became glaucomatous; an outcome depending on the fact that in these cases the abnormal stretching of the suspensory ligament is absent. Such lenses sooner or later become opaque and degenerated but do not become completely absorbed. The vitreous is more or less fluid and contains fine opacities, otherwise the eyes show very little reaction and vision, on the average, remains good. in view of these considerations the author suggests reclination in cases of secondary glaucoma due to subluxation. Dislocation into the anterior chamber, whether traumatic or spontaneous is very dangerous. Both inflammatory and mechanical complications occur. In 16 out of 17 cases observed by Hegner there was glaucoma, in the seventeenth an early extraction had been done. Extraction, however, is dangerous here also; it was done in 13 out

of the 16 cases, five eyes being lost and useful vision being obtained in only three cases. Lenses in the anterior chamber undergo degenerative changes more rapidly than in other forms of dislocation.

Thirteen cases of congenital and spontaneous dislocation were followed up. They may be divided into two groups; those in which the suspensory ligament is intact and those in which its development is defective. The former group has a relatively good prognosis as neither total luxation nor glaucoma tends to occur. The second group tends to produce complete dislocation, but not glaucoma. To this group belongs a rare condition, familial spontaneous dislocation. Finally, when the lens becomes dislocated in association with an already existing ocular disease such as high myopia or hydrophthalmos the prognosis is bad.

H. M. TRAQUAIR.

(2) **Marin (Almeira).—Traumatic subconjunctival dislocation of the lens with intact iris. (Luxation traumatic subconjunctival del cristalino con emigration del mismo e integridad del iris.)** *Archivos de Oftalmologia Hispano-Americanos*, July, 1917.

(2) **Marin** records the rare result of an ocular injury in a man of 75 years of age. The left globe was ruptured in the upper and outer part, the lens was lying outside, under the conjunctiva, and the iris was uninjured. The vitreous was full of blood. As soon as the wound in the sclera was healed, the dislocated lens was removed through a conjunctival incision. The vitreous haemorrhage cleared up under the use of dionin, allowing a view of the fundus to be obtained. The resulting vision was finger counting at one metre.

R. R. JAMES.

(3) **Paine, Howard S. (Glen Falls, N.Y.) — Operation for extraction of lenses fully dislocated into the vitreous.** *Amer. Jl. of Ophthal.*, Series 3, Vol. I, No. 3, March, 1918.

(3) A large part of **Paine's** article is taken up with quotations from distinguished surgeons to the effect that lenses dislocated into the vitreous are inoperable and best left alone. Paine holds that such cases are not always desperate. In operating, the great and vital necessity is to see what one is doing, i.e., to see the lens itself. In order to do this the operation is carried out in artificial light, arrangements being made to illuminate the interior of the eye very thoroughly. The surgeon sits at the patient's head and, after the incision has been made, is able to look down into the depths of the vitreous and see what he is doing. Personally the author wears a

broad high shade and assists himself with spectacles stronger than those ordinarily worn. He says that Reber recommended "as an operating glass, a sphere with a plus four degree prism, base in." This possibly is a printer's error, for "plus sphere with a four degree prism." In operating Paine passes a Stevens traction hook to the lowest border of the lens, lifting or drawing it into the pupil, then slipping a Smith spatula behind it and sliding it up and out on the spatula by traction on the hook if it holds, or gentle pressure on the outside of the cornea if the hook pulls out." Five successful operations are recorded. It is obvious that the essential point according to Paine is the illumination of the interior of the eye. The exact details of the position and handling of the light do not seem to be given. It is said that the globe should be frosted, with a corkscrew filament, and of about 20 candle power, and a condensing lens is employed. This article is well worth study, but it is the curious mixture of the personal experience and that of others which makes it a little difficult to read.

ERNEST THOMSON.

III.—OCULAR SYPHILIS

(1) **Alter, Francis W.—Palpebral syphilis, a review with case report.** *Ophthalmology*, January, 1916.

(1) According to **Alter**, of Toledo, Ohio, a comparison and averaging of the statistics of various observers shows that chancres of the eyelids constitute about three to five per cent. of extragenital chancres. The lesion has some of the characters both of chalazion and hordeolum, but there is a nibbled, punched-out area at the lid margin. The base is indurated, and indolence and chronicity are characteristic. There is enlargement of the preauricular or submaxillary gland. The finding of the spirochaete and a positive Wassermann reaction will help the diagnosis. The lesion seldom develops on the outer skin surface of the lid. It is almost invariably on the actual lid margin, in the canthi, or on the conjunctiva tarsi. It may be accompanied by pannus.

The secondary syphilitic skin eruptions do not affect the lids much, if at all, though mucous patches may occur on the conjunctiva. The most common tertiary lesion is tarsitis. It gives rise to a peculiar form of vascular hypertrophy of the lid, with ptosis.

A. J. BALLANTYNE.

(2) **Charles, J. W. (St. Louis).—Dental stigmata of syphilis and transillumination in the demonstration of Hutchinson's teeth.** *Amer. Jl. of Ophthal.*, March, 1917.

(2) In the first four pages of this article **Charles** is mainly concerned with the pathology, from the dental standpoint, of Hutchinson's teeth, and quotes extensively from dental writers. In the last page he describes how, in connection with his work at the Missouri School for the Blind, he had noticed several cases of Hutchinson's teeth in which only one central incisor was notched, and in which he sought evidence of malnutrition in the teeth which had not, in ordinary light, exhibited any abnormality. By placing an electric light behind the incisors it was found in several cases of suspected syphilis not only that the companion tooth of a notched incisor was affected by a disturbance in enamelification of such a kind that the normal enamel could be distinguished from the abnormal, but also that occasionally there was a case, in which no notching had appeared in either tooth, which showed this area of deficient enamel. The reviewer feels that this is an important contribution to our knowledge and that the author's summary of the dental pathology of these cases should be read in the original by those—possibly most of us—whose knowledge of dental pathology is mainly limited to certain painful experiences.

ERNEST THOMSON.

(3) **Marbaix (Tournai). What is the correct attitude to take up where there is mydriasis in an old syphilitic case without any other apparent manifestation? (Quelle attitude prendre en présence d'une mydriase chez un syphilitique ancien sans autre manifestation apparente.)** *La Clin. Ophtal.*, November, 1919.

(3) What the author seems to mean is this. Is the mydriasis merely a local sign of a local manifestation of syphilis or is it to be regarded as the warning of the onset of tabes or general paralysis? In the former case it may be possible to do nothing. In the latter treatment is essential. Assuming that the blood Wassermann is negative that does not justify abstention from treatment nor the authorization of marriage until the cerebro-spinal fluid has been examined. One's conduct will be guided by the plus or minus result. If the result is positive and indicative of a meningitis then intensive treatment must be undertaken to arrest or retard the onset of general paralysis or tabes. If negative, "abstention (from treatment) is perhaps defensible, the pupillary lesions then indicating a very localised syphilis." Obviously, if the blood Wassermann is positive there is no question as to abstention from treatment.

ERNEST THOMSON.

(4) **Lakaye, Dr. R. (Liège).—Early post-arsenical syphilitic nervous manifestations. (Les manifestations syphilitiques nerveuses précoces postarsenicales.)** *Arch. méd. belges*, July, 1920.

(4) **Lakaye** draws attention to the numerous cases of early cranial nerve paralysis in syphilis treated by 606. Such paralyses occur suddenly two or three months after the termination of the first course of treatment and are sometimes preceded by general symptoms such as headache and prostration. Lumbar puncture at this time gives evidence of abundant leucocytosis with albuminosis, indicating a cerebral and spinal meningitis, chiefly basilar. Such meningitis in the secondary period was known to be of frequent occurrence, but did not, in the absence of arsenical treatment, generally give rise to any serious symptoms. Lakaye considers that this treatment renders the central nervous system less resistant to an affection it is otherwise capable of resisting. He quotes illustrative cases in support of his contention that it is at the outset of secondary symptoms that these accidents occur and in the presence of a negative blood Wassermann reaction. He points out that the best way of avoiding these accidents is to combine arsenical treatment with mercurial injections either simultaneously or alternately, and prefers the more soluble preparations such as the cyanide, biniodide or benzoate.

In treating the paralyses he continues the arsenical treatment in small doses very slowly increased, but not beyond 60 ctgs. At the same time he administers every week an intramuscular injection o 10 ctgs. of salicylate of mercury or of calomel. The result has always been successful except in some cases of facial paralysis in which slight atrophy of the muscles has remained.

The paper is not written for ophthalmic surgeons, but so many of the cases recorded affected ocular nerves as to render it of some interest to our readers.

E.E.H.

(5) **Darier, A. (Paris).—The diagnostic importance of heredo-specific teeth. (Importance diagnostique des dents hérédo-spécifiques.)** *La Clin. Ophtal.*, September, 1922.

(5) **Darier** has before alluded to the importance of the first permanent molar as a tooth which, as well as the incisors, may be affected in hereditary syphilis. He returns in this article to the subject which will be found discussed in full in his "Traité complet de Thérapeutique oculaire," published in 1921, and reproduces the diagram which will be found on page 25 of that work. The enamel or gum of the first permanent molar ossifies one month before birth, those of the median incisors in the first month of life,

and those of the lateral incisors and canines in the fourth and fifth months of life. The importance of these facts is obvious when one is considering a case of hereditary syphilis, since, according to whether one or other or all three of these dental groups are affected one can form a judgment as to the era of incidence of the disease. The drawing shows the usual notch in the incisors, the single bare peg of dentine in the canines and the four bare pegs of dentine in the first permanent molar.

ERNEST THOMSON.

(6) **Gourfein. D. (Geneva).—The efficacy of bismuth salts in the treatment of ocular affections of syphilitic origin. (L'efficacité des sels de bismuth dans le traitement des affections oculaires d'origine syphilitique.)** *Rev. Gén. d'Ophtal.*, April, 1923.

(6) **Gourfein** continues his observations on intra-muscular injections of tartro-bismuthate of potassium and sodium, the first notes on which were published by him in the *Revue Génerale d'Ophtalmologie* for January, 1922, and were abstracted in this Journal for June, 1923, *q.v.* Gourfein has now treated 33 cases of syphilitic eye disease and reports nine of them in detail. Of these nine, three were cases of iridocyclitis, four of paralysis of muscles, and two of choroido-retinitis. The results have been very satisfactory, and the author concludes that bismuth is a first class "treponemicide," though he admits that its effect upon the serum-reaction is less rapid than upon the symptoms of the disease. Reference to the abstract of his previous article above referred to will show that at that time Gourfein said "The future only can show whether this treatment realizes the much-wished-for "*therapia sterilans magna.*" Apparently it does not, at any rate in some cases. The conclusion come to is that treatment by the salts of bismuth presents great advantages. It is active, and rapidly cures syphilitic ocular "accidents." It frequently sterilizes the organism. It is well tolerated both by adults and children. It does not expose the patient to grave optic nerve complications nor to other nervous complications like serous apoplexy. In short it does not present the possibility of danger or inconvenience as does arsenobenzol medication. Both these articles will repay perusal.

ERNEST THOMSON.

IV.—TRAUMATIC AFFECTIONS OF RETINA

(1) **Stähli (Zürich).—Contribution to our knowledge of traumatic angiopathy of the retina.** (**Zur Kenntnis der Angiopathia retinae traumatica.**) *Klin. Monatsbl. f. Augenheilk.*, Sept.-Oct, 1915.

(1) Traumatic retinal angiopathy was described by Purtscher in 1910. Following severe injury to the skull white spots and haemorrhages occur in one or both eyes in the area of the nerve entrance and macula.

Stähli's patient fell off a vehicle injuring the right occipital region and suffering many other minor injuries. Beyond some blood in the conjunctivae there was no other sign of cranial injury. A good recovery was made in every way but the vision of the right eye was reduced and that of the left almost destroyed. A month after the accident both fundi showed numerous and extensive white patches and small haemorrhages. The lesions were more pronounced in the left eye. In other respects the eyes were normal. Some recovery of vision took place and the fundus changes diminished. A year later the fundus changes had practically disappeared but both optic nerves were pale especially on the temporal sides. Useful vision was absent in the left eye owing to a large central scotoma but the right eye could read 5/6 with difficulty. Stähli regards this condition as closely related both in causation and in nature to Berlin's opacity in commotio retinae.

H. M. TRAQUAIR.

(2) **Francisco, M. Fernandez.—Traumatic retinal detachment in both eyes.** (**Desprendimiento de la Retina en ambos ojos de origen traumatico.**) *Cronica Medico Quirurgica de la Habana*, December, 1918.

(2) The author records the case of a man who received a severe injury to the head and one arm in a riding accident. The patient was badly concussed and was unconscious for ten days; on regaining consciousness he complained of a defect of sight in each eye. Ophthalmoscopic examination revealed in each a transparent detachment of the retina, extending up to the disc and involving the macula.

The treatment adopted was purely medical, absolute rest in bed, nux vomica internally, sweating by means of pilocarpin injections, and, locally, dionin and heat. After four months treatment the man had completely recovered.

R. R. JAMES.

(3) **Lacroix.—Traumatic detachment of a retinal vein. (Décollement traumatique d'une veine rétinienne.)** *Arch. d'Ophtal.*, Jan.-Feb., 1919.

(3) The patient, a soldier, was struck by a splinter of wood inflicting a non-penetrating wound of the left upper lid close to the inner canthus. When admitted two days later vision was hand-movement at 10 cm. There was no external lesion of the eyeball. Dust-like opacity of the vitreous obscured the fundus. Six days later the fundus was visible and choroido-retinal ruptures were discovered close to the O.D. After another five days' interval the vitreous was almost clear and a large choroido-retinal rupture in the macular region was discovered. Starting from near the upper border of the disc a long narrow rupture passed obliquely across the fundus and extended towards the ora serrata.

The inferior temporal vein, after running its usual course for a short distance, suddenly left the retinal level and protruded anteriorly. The displacement of the vessel was considerable; it was free in the vitreous, and was obviously attached at the periphery, as its contour and calibre were normal and its blood stream uninterrupted. The subjacent retina was not detached and showed no breach of continuity, though the colour of the fundus at this part was paler than elsewhere. No modification of these appearances occurred during the month following the injury. The man was then evacuated.

J. B. LAWFORD.

(4) **Frenkel, Henri (Toulouse).—A new case of folding of the retina in circles due to contusion of the posterior segment. (Un nouveau cas de plissement en cercles de la rétine par contusion du segment postérieure.)** *La Rev. Gén. d'Ophtal.*, Janvier, 1920.

(4) We welcome the reappearance of this Journal, the publication of which was in abeyance during the war.

Frenkel in 1917 published a case of multiple detachments of the retina in circles which was then considered unique, but the same author has come across a second case almost exactly the same, and due, in the author's opinion, to the same cause, namely, contusion due to windage of a shell. The case is described in detail and a plate shows the detachments very well. The first case has been seen again and was found to present exactly the same lesions as when first examined in 1916. In addition the disc was found to be cupped and atrophic and the tension plus 1. The present case was first examined in 1918, though the accident of exposure to shell windage occurred as far back as 1915. The author concludes that this form of retinal detachment has a tendency to remain stationary. He does not feel disposed to risk a hypothesis as to the chronic glaucomatous condition which has supervened in the first case.

ERNEST THOMSON.

OBITUARY

Thomas Pridgin Teale, F.R.S.

THOMAS PRIDGIN TEALE, junior, came from an old medical stock, his father Thomas Pridgin Teale who died in 1867, having been for many years an eminent surgeon on the old Infirmary staff, and F.R.S. and the inventor of the well-known Teale's amputation. Thomas Pridgin Teale, junior, was born in 1831, educated at the Leeds Grammar School and Winchester College. From Winchester he went to Brasenose College, Oxford, where he took his degree in 1852 in mathematics with high honours. He then became a medical student at King's College, where he worked under Fergusson and Bowman. Later Sir William Bowman appointed him his assistant at Moorfields Eye Hospital. After taking the M.R.C.S.Eng., in 1855, he spent six months on the Continent, chiefly in Paris with Liebreich, where he became familiar with the use of the newly invented ophthalmoscope. On returning home he took the M.A. and M.B., Oxford, and the F.R.C.S. Eng., in 1857. He became Demonstrator of Anatomy and then Lecturer in that subject at the Leeds School of Medicine, and subsequently Lecturer in Surgery, 1856-1876. In 1864, he wa appointed Surgeon to the General Infirmary at Leeds, holding office for 20 years, when he became Consulting Surgeon with six beds and these he used for another 20 years when he retired from practice.

As his hospital appointment did not embrace ophthalmology he established a clinique in rooms behind his house in Park Row for this special work and continued it for several years. He was a Crown member of the General Medical Council from 1876 to 1901. On the establishment of the University of Leeds in 1904 he received the honorary degree of D.Sc. He was an active member of the Board of the Leeds Infirmary from 1864 to 1904, rarely attending meetings after that time.

When Pridgin Teale settled in Leeds he intended to confine himself to ophthalmic work, but before he had established himself in this branch he was drawn off to assist his father in his large surgical practice, and so he came to combine surgery and ophthalmology.

Though he was not a prolific writer, he was an enthusiastic and excellent teacher sparing neither time nor effort in pointing out to his students the various phases of diseases and their treatment, illustrating his remarks whenever possible by drawings and diagrams, upon the value of which as an aid to memory he was most insistent.

His own case books are full of figures and diagrams rapidly drawn in coloured chalks, accompanied by a few concise remarks. Small in stature, and delicately built, he had beautiful hands—hands made for an operator. His ophthalmic operations were well planned, swiftly and neatly executed with great attention to detail.

Among his most important early writings were papers on the use of mercury inunction in iritis; bulb-shaped probes in lacrimal obstruction; extraction of soft cataract by suction, an operation which he practised most successfully using a suction curette of his own devising. All through his life he was a steadfast upholder of simple extraction of cataract without iridectomy making the corneal incision midway between the upper corneal margin and the upper level of the pupil. In 1893, he delivered the Bowman Lecture with this as his thesis. At the meeting of the British Medical Association in Leeds in 1889 he delivered the address in Surgery on "Detail in Surgery."

Teale was a great sanitarian and a pioneer in house sanitation and was for many years President of the Yorkshire Association of Sanitary Inspectors upon whom his high ideals had a very marked influence. Months of experiments and research into the economic aspects of coal combustion resulted in the now well-known Teale "Fire Grate" (since somewhat modified by his eldest son) which created a revolution in the design of the old wasteful fire grate, and a paper on this subject in 1888 procured for him the Fellowship of the Royal Society.

Enthusiasm, simplicity, and gentleness of character were the outstanding features of a charming personality. Teale's one idea in his professional work was the good of his patient and to that he gave all his thought and energy. Money he never troubled about. Reputation for himself he never studied—the only reputation he cared for was that of his profession or of an operation which he knew to be valuable.

To the very last his enthusiasm was remarkable; he was keenly interested in everything about him. Fishing was his one great hobby and recreation. He will be greatly missed in Leeds and Yorkshire where his kindly interest and sympathy in every form of social service was never failing.

H. SECKER WALKER.

CORRESPONDENCE

CASE OF IMPLANTATION CYST OF IRIS AND CILIARY BODY

To the Editor of THE BRITISH JOURNAL OF OPHTHALMOLOGY

SIR,—The interesting case of "Implantation Cyst of Iris and Ciliary Body" in your issue of November, communicated by Dr. Barrie Brownlee, gives one to think.

It no doubt resulted from epithelial cells being carried into the

globe by means of a too deeply inserted sature during the operation of advancement, and the writer comments on the frequency with which the sclera has been completely perforated by the needle during such operations.

I am sure I have done it in the past, and have had many an anxious moment wondering if I had done any serious injury to the globe, always having in my memory the account of a young lady, who, very sensitive about her appearance owing to a disfiguring squint, went to London, unknown to her parents, for operative treatment at the hands of an ophthalmic surgeon, who, unfortunately inserted a suture too deeply, with the result that the eye became infected and had to be removed.

The odd thing, to my mind is, that so seldom any harm results; rarely does an eye become septic after an operation for squint nowadays, and an implantation cyst is rare indeed.

But, thanks largely to Bishop, of Chicago, any risk of inserting needles too deeply into the sclera and of also causing implantation cysts has been entirely abolished by, what I have found, a far better and more accurate method.

In the old advancement operation, besides the above risks, there was a good deal of guessing, one cut off a variable length of the tendon and one stitched the remainder forward, so as to produce a result as near perfection as one could, but that result was often very uncertain, and one wished that one had cut off a little more or a little less of the tendon, or had inserted the sutures into it a little further back or not quite so far.

When it came to removing those sutures a few days later, it was not always easy for the surgeon or pleasant for the patient in spite of the assistance of cocain with adrenalin. But tendon tucking, in lieu of advancement, is, with Bishop's instrument, another thing.

It is a most fascinating piece of mechanism, one sees quite accurately what one is doing and the amount thereof, and, the more I use it, the more satisfied and charmed I am with it. The rectus tendon should be thoroughly exposed and defined, and care should be taken that the "prongs" of the tucker only take up the tendon and do not catch in any of the neighbouring tissue, or there may be a little difficulty in detaching it at the end of the operation.

I am greatly indebted to Col. Elliot—always ready to help the novice—for my knowledge of the method; he not only gave me valuable hints about it, but actually lent me the "tucker" he had brought from America to try; he also made the valuable suggestion that, instead of tying the tuck above and below with catgut, one should use buried silk sutures for the purpose, and, having tried both, I now always use silk, and find I can tie up the tuck much more closely and firmly therewith than I could with catgut.

Col. Elliot made another valuable suggestion that the silk sutures in the tuck should be touched with tincture of iodine to render the assurance against septicity doubly sure.

I have followed his advice in this particular also, except in one case, in which I entirely forgot it, with the result that it did no whit worse than the others.

I have so far this year done eleven tuckings, all in private. In the last thirty-four years, though I have done many advancements, I cannot say I ever really liked the operation, and I have many times disliked it. Tendon tucking with Bishop's instrument is very fascinating.

Yours faithfully,
S. JOHNSON TAYLOR.

NORWICH,
Nov. 9th, 1923.

NOTES

Death

WE regret to announce the death, on November 15, at Ellerslie, Chichester, the residence of his sister, of GEORGE THOMAS BROOKSBANK JAMES, of London and Otterburn, Eastbourne, son of the late William James, of Otterburn Tower, Northumberland, aged 56 years. The deceased gained the entrance scholarship to Westminster Hospital and took the F.R.C.S. in 1896. He was Surgeon to the Royal Eye Hospital, Southwark, and Ophthalmic Surgeon to Westminster Hospital. He held the posts of Honorary Ophthalmic Surgeon, Princess Alice Memorial Hospital, Eastbourne and Senior Clinical Assistant at the Royal Westminster Ophthalmic Hospital. He was author of "Addenda to Descriptive Catalogue of Westminster Hospital Museum," 1898-99 and joint author for 1900; "Three Cases of Juvenile Tabes"; "On the Measurement of the Stereoscopic Visual Acuity"; "The Treatment of Gonorrhoeal Conjunctivitis in the Adult" (with J. Stroud Hosford); "The Operative Treatment of Strabismus"; and "The Operative Treatment of Glaucoma: a new Method."

* * * * *

Appointments

DR. WALTER B. LANCASTER has been elected President of the American Academy of Ophthalmology and Oto-Laryngology,

H. SUGDEN VIVIAN has been appointed Honorary Ophthalmic Surgeon to the Grimsby and District Hospital, and Ophthalmic Surgeon to Grimsby Board of Guardians.

Dr. J. G. DE FOCK, Senior Ophthalmic Surgeon, Johannesburg Hospital, has been appointed Lecturer in Ophthalmology, Witwatersrand University.

* * * * *

Ophthalmological Society of Egypt

THE Ophthalmological Society of Egypt has elected the following officers for 1923-24:—President: Dr. Mahmoud Zaky; Vice-President: Dr. Zaki Seddik; Hon. Secretary: Dr. Hassan Barrada; Assistant Hon. Secretary: Dr. Galal Abul Seoud; Hon. Treasurer and Archivist: Dr. A. F. MacCallan.

The Annual Meeting of the Ophthalmological Society of Egypt will take place at the School of Medicine Examination Room, Student's Club, Cairo, on March 7th, 1924.

* * * * *

Sir George Andreas Berry and Parliament

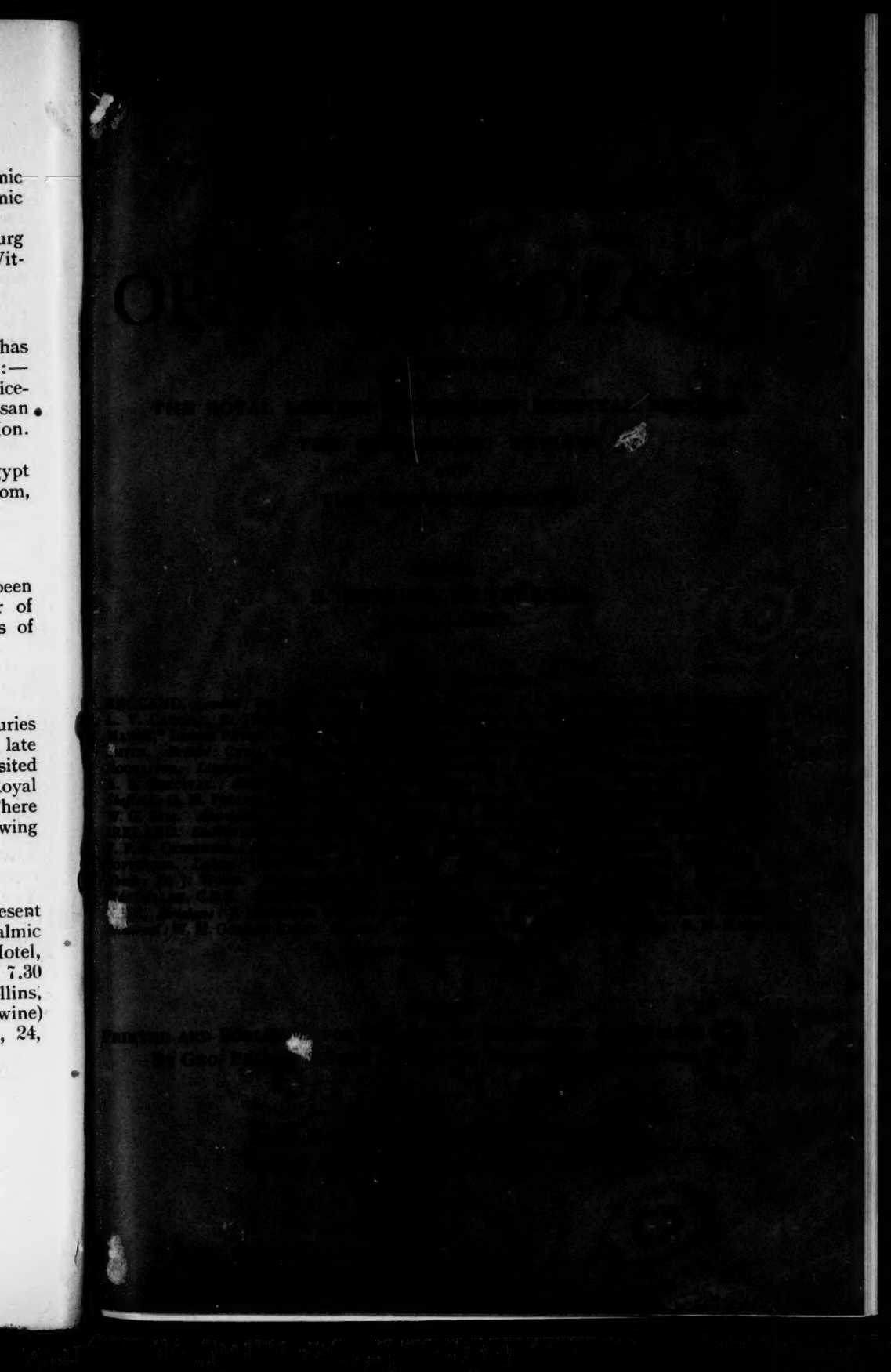
SIR GEORGE ANDREAS BERRY has been returned unopposed as Unionist Member of Parliament for the combined Universities of Edinburgh and St. Andrew's.

* * * * *

Collection of Illustrations of War Injuries of the Fundus at the Royal College of Surgeons of England

THE Collection of illustrations of War Injuries of the Fundus, made by Dr. WALLACE, late temporary Captain R.A.M.C., is now deposited in the Army Medical War Museum of the Royal College of Surgeons of England and is available for study. There is a descriptive catalogue, and each drawing has a diagram showing the direction of the projectile which caused the wound.

* * * * *

Royal London Ophthalmic Hospital

THE Annual Dinner of past and present students of the Royal London Ophthalmic Hospital will be held at the Langham Hotel, Portland Place, on Thursday, February 7, 1924, at 7 for 7.30 o'clock under the chairmanship of Mr. E. Treacher Collins, Consulting Surgeon to the Hospital. Tickets (excluding wine) 15s.; applications should be made to Sir William Lister, 24, Devonshire Place, W.1.